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L4             0 S L3 AND LIVER?
L5          9494 S (FATTY ACID BINDING PROTEIN)
L6          1324 S L5 AND PLASMA?
L7           397 S L6 AND LIVER?
L8            21 S L7 AND REVIEW?
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L11            5 DUPLICATE REMOVE L10 (9 DUPLICATES REMOVED)
L12          351 S (LIVER FABP)
L13           23 S L12 AND PLASMA?
L14           10 DUPLICATE REMOVE L13 (13 DUPLICATES REMOVED)

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ANSWER 8 OF 10 CAPLUS COPYRIGHT 2004 ACS on STN
AN 1991:226433 CAPLUS
DN 114:226433
ED Entered STN: 15 Jun 1991
TI Intracellular sterol distribution in transfected mouse L-cell fibroblasts expressing rat liver fatty acid-binding protein
AU Jefferson, John R.; Slotte, J. Peter; Nemecz, Gyorgy; Pastuszyn, Andrzej; Scallen, Terence J.; Schroeder, Friedhelm
CS Med. Cent., Univ. Cincinnati, Cincinnati, OH, 45267-0004, USA
SO Journal of Biological Chemistry (1991), 266(9), 5486-96
CODEN: JBCHA3; ISSN: 0021-9258
DT Journal
LA English
CC 13-2 (Mammalian Biochemistry)
AB The potential role of liver fatty acid-binding protein (L-FABP) in modulating cellular sterol distribution was examined in mouse L-cell fibroblasts transfected with cDNA encoding L-FABP. L-cells were chosen because they contain only a small amount of endogenous FABP which does not bind [3H]cholesterol, does not enhance intermembrane sterol transfer, and whose content is unaltered by the expression of L-FABP. Transfected L-cells expressed 0.34% of cytosolic protein as L-FABP. Transfection alone with low expression of L-FABP (0.008% of cytosolic protein) had no effect on any of the parameters tested. Three aspects of cellular sterol transfer were examined First, cellular sterol uptake, monitored by [3H]cholesterol and the fluorescent sterol, Δ-5-,7,9(11),22-ergostatetraen-3β-ol, was increased 21.5% in L-cells expressing L-FABP. This increase was not accounted for by increased sterol esterification in the cells expressing L-FABP. Inhibition of both cholesterol transfer and esterification with 3-(decyldimethylsilyl)-N-[2-(4-methylphenyl)-1-phenylethyl]propanamide from Sandoz abolished the L-FABP-related enhancement of both [3H]cholesterol uptake and esterification. Second, **plasma** membrane transbilayer distribution of sterol, determined by fluorescence methods indicated that the majority of sterol was in the inner leaflet of the **plasma** membrane. In transfected cells expressing L-FABP, twice as much sterol (28%) was present in the exofacial leaflet of the **plasma** membrane as compared to that of control cells (15%). Third, expression of L-FABP enhanced sterol transfer from the **plasma** membrane to microsomes in intact cells. Treatment of [3H]cholesterol or [3H]oleate-loaded cells with sphingomyelinase resulted in increased formation of radiolabeled cholesterol ester, consistent with enhanced microsomal esterification of **plasma** membrane-derived cholesterol. Concomitantly, **plasma** membrane [3H]cholesterol became less accessible to oxidation by cholesterol oxidase. Sphingomyelinase-stimulated cholesterol esterification was 21% greater in transfected cells. Concomitantly, accessibility of **plasma** membrane [3H]cholesterol to cholesterol oxidase was decreased 18% in cells expressing L-FABP. These differences are consistent with the ability of L-FABP to influence sterol transport and **plasma** membrane transbilayer sterol distribution in intact cells.
ST sterol fatty acid binding protein; membrane cholesterol **liver FABP** protein
IT Sphingomyelins
RL: BIOL (Biological study)
(of cell membrane, L-FABP effect on, sterol content in relation to)
IT Cell membrane
Endoplasmic reticulum
(sterols in, L-FABP modulation of)
IT Proteins, specific or class
RL: BIOL (Biological study)
(L-FABP (liver fatty acid-binding protein), sterol distribution in cells regulation by)

IT Biological transport
(absorption, of sterols, L-FABP expression effect on)
IT Cytoplasm
(cytosol, L-FABP protein in, intracellular sterol distribution regulation by)
IT Steroids, biological studies
RL: BIOL (Biological study)
(hydroxy, intracellular distribution of, L-FABP protein regulation of)
IT 9027-63-8
RL: BIOL (Biological study)
(L-FABP stimulation of, intracellular sterol distribution in relation to)
IT 57-88-5D, Cholesterol, esters
RL: FORM (Formation, nonpreparative)
(formation of, from free cholesterol, L-FABP protein regulation of)
IT 516-85-8, Dehydroergosterol
RL: BIOL (Biological study)
(intracellular distribution of, L-FABP protein modulation of)
IT 57-88-5, Cholesterol, biological studies
RL: BIOL (Biological study)
(intracellular distribution of, L-FABP protein regulation of)

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AN 2003:81756 BIOSIS

DN PREV200300081756

TI **Plasma** concentration of intestinal- and **liver**-**FABP** in neonates suffering from necrotizing enterocolitis and in healthy preterm neonates.

AU Guthmann, Florian [Reprint Author]; Boerchers, Torsten; Wolfrum, Christian; Wustrack, Thomas; Bartholomaeus, Sabine; Spener, Friedrich

CS Department of Neonatology, Charite Campus Mitte, D-10098, Berlin, Germany florian.guthmann@charite.de

SO Molecular and Cellular Biochemistry, (October 2002) Vol. 239, No. 1-2, pp. 227-234. print.
ISSN: 0300-8177 (ISSN print).

DT Article

LA English

ED Entered STN: 6 Feb 2003
Last Updated on STN: 6 Feb 2003

AB Both early diagnostic and prognostic assessment of the acute abdomen in preterm infants are hampered by the lack of a sensitive and specific parameter for intestinal injury. In this prospective clinical study we wanted to estimate the value of intestinal (I-) and liver (L-) fatty acid binding protein (FABP) in diagnosing necrotizing enterocolitis (NEC). Using highly sensitive and specific sandwich ELISAs which employ recombinant human I- and L-FABP as standard proteins (limit of detection 0.1 ng/ml **plasma**), the L-FABP concentration (median 7.6 ng/ml) was determined to be about 3 fold that of I-FABP (median 2.52 ng/ml) in **plasma** of healthy preterm infants. I- and L-FABP concentrations significantly increased with birth weight (1.6 and 5.0 ng/ml per kg, respectively). At onset of symptoms, I-FABP concentration was significantly higher in infants who later developed severe NEC compared to healthy infants and those, whose illness remained confined to stage I or II. L-FABP was significantly elevated compared to the control group at onset of symptoms regardless of the further course of NEC. In conclusion, I-FABP appears to be a specific parameter for early detection of intestinal injury leading to severe NEC stage III. L-FABP, however, is a promising sensitive marker even for stage I of NEC.

CC Pathology - Diagnostic 12504
Digestive system - Physiology and biochemistry 14004
Digestive system - Pathology 14006
Blood - Blood and lymph studies 15002
Blood - Blood cell studies 15004
Pediatrics 25000
Medical and clinical microbiology - Bacteriology 36002

IT Major Concepts
Gastroenterology (Human Medicine, Medical Sciences); Infection; Pediatrics (Human Medicine, Medical Sciences)

IT Parts, Structures, & Systems of Organisms
intestine: digestive system; **plasma**: blood and lymphatics

IT Diseases
necrotizing enterocolitis: bacterial disease, digestive system disease, diagnosis
Enterocolitis, Necrotizing (MeSH)

IT Chemicals & Biochemicals
intestinal-fatty acid binding protein

ORGN Classifier
Hominidae 86215
Super Taxa
Primates; Mammalia; Vertebrata; Chordata; Animalia
Organism Name
human (common): newborn, premature
Taxa Notes

ANSWER 1 OF 10 BIOSIS COPYRIGHT (c) 2004 The Thomson Corporation. on STN DUPLICATE 1

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Enterocolitis, Necrotizing (MeSH)

IT Chemicals & Biochemicals
intestinal-fatty acid binding protein

ORGN Classifier
Hominidae 86215
Super Taxa
Primates; Mammalia; Vertebrata; Chordata; Animalia
Organism Name
human (common): newborn, premature
Taxa Notes

Animals, Chordates, Humans, Mammals, Primates, Vertebrates

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